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FUNCTIONAL HEART-BLOCK.[1]

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The factors involved in the coördination of the activities of the auricles and the ventricles have for a long time been subjects of investigation by physiologists, clinicians, and pathologists. The many skilfully devised experiments, clinical observations, and autopsy findings have furnished much valuable material upon which our present conceptions are based. That destruction of the A-V bundle results in the complete dissociation of auricular and ventricular activity seems well established. The effect of insults to the A-V bundle of a degree short of complete destruction, the relative importance of the muscle and nerve elements of the A-V junction, the changes in the function of the junctional tissues brought about by factors other than organic lesions, and the influence of the nerves on the independent activities of the ventricles, are some of the questions still open to investigation.

It is with the hope of presenting some clinical facts which have a bearing on the elucidation of these problems that the cases of this paper are reported.

We have applied the term "functional heart-block" to the group of cases here presented not because we believe organic changes are absent, but because these changes are of such a moderate degree or are of such a nature that by the administration of drugs the evidence of functional abnormalities can be considerably modified. The evidence that digitalis and atropin greatly modify the cardiac activity in these cases is quite clear. If we accept the myogenic theory of cardiac activity these cases present a considerable field for interesting speculation. This theory has considerable evidence on which to base its assumption that the property of stimulus conduction is dependent on the formation of definite chemical molecules in the muscle cell, and that with each passage of a stimulus these molecules are disrupted, thus producing a refractory phase which continues until a sufficient time has elapsed to allow these molecules to reform; it is further believed that external nerve influences may have a modifying effect in changing the rapidity of the construction of these molecules. Hence one may ask in such cases as these here presented, Do digitalis and atropin act by a direct influence on the muscle cell or is their effect obtained by producing a change in the activity of the vagus which causes a change in the balance of the influences brought to the muscle cell by the vagi and sympathetics?

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[1] Read at the meeting of the American Society for the Advancement of Clinical Investigation, Atlantic City, May 11, 1914.

It has been our privilege to study more or less completely thirty-four cases of heart-block presenting great diversities in clinical features. The following cases have been selected, since they represent certain types; some of our remarks, however, are based on observations on cases not reported in detail but which present aspects conforming to the types here set forth.

CASE I.—*Decompensated mitral insufficiency; digitalis heart-block promptly relieved by atropin.*

L. S., aged sixty-two years, tailor; a native of Russia. Admitted to the Presbyterian Hospital in November, 1913. His chief complaint was dyspnea and swollen legs. The patient had been accustomed to drink six to eight glasses of beer and one whisky a day and smoked six to eight small cigars a day. At nineteen years of age he had gonorrhea and chancre (?), which were treated for two weeks, with complete recovery and no subsequent symptoms. He had never had rheumatism, and had been perfectly free from illness except as above until five years before admission; at this time he felt weak and was examined by a physician, who told him he had "heart disease" and must stop work. Notwithstanding this advice he continued his work until a few days before his admission to the hospital. For three years he has had considerable dyspnea on exertion and swollen legs. For five days preceding his admission he had been taking a half-ounce of infusion of digitalis three times a day.

Physical Examination (November 19). A large man, somewhat obese; propped up in bed; no cyanosis; some dyspnea; respiration 26. There were a few rales at both bases of the lungs. The cardiac dulness was found abnormally increased both to right and left.

The heart sounds were indistinct at the apex and at the base. A loud, blowing, systolic murmur was heard all over the precordium. The pulse was irregular and changed in the course of a few minutes from a rate of 44 to 78; all the heart impulses were transmitted to the wrist; all the radial beats in a short period of time were apparently of equal force. No interpolated heart sounds could be detected during the periods of slow rate; after the exertion of sitting up in bed the pulse rate rose to 86. There was moderate edema of the legs.

Laboratory Findings. The urine output averaged about one liter per twenty-four hours during his stay in the hospital and constantly showed a trace of albumin and a few casts. A functional kidney test with sulphophenolphthalein showed an output of 47 per cent. in two hours; the dye first appeared in the urine ten minutes after its injection.

The blood showed nothing abnormal; the Wassermann reaction was negative.

On the next morning the heart was beating somewhat irregularly at a rate of 40 to 50 a minute and showed no tendency to change to a more rapid rate on exertion. At 10.25 A.M., an electrocardiogram was taken which showed complete block. At 10.30 A.M. $\frac{1}{50}$ grain of atropin was administered hypodermically. Observations of the heart rate showed the following:

November 20, A.M.	Pulse somewhat irregular rate 40 to 50.
10.25 A.M.	Record taken, As = 80; Vs = 46; complete block.
10.30 A.M.	Atropin, grain $\frac{1}{50}$ hypodermically.
10.35 A.M.	Vs = 48.
10.40 A.M.	Vs = 48.
10.45 A.M.	Vs = 48.
10.50 A.M.	Vs = 50.
10.51 A.M.	Record (Fig. 1) taken, As = 85, Vs = 46, complete block.
10.56 A.M.	Record (Fig. 2) taken, As = 85, Vs = 85, P – R = 0.4 seconds.
11.01 A.M.	Record (Fig. 3) taken, As = 85, Vs = 85, P – R = 0.22 seconds.
11.06 A.M.	Record (Fig. 4) taken, As = 80, Vs = 80, P – R = 0.2 seconds.

The electrocardiographic records of this case here reproduced were all taken by Lead I (right arm and left arm). The record taken at 10.25 A.M. is not shown, since it is identical in form with the record taken at 10.51 A.M. (Fig. 1), which shows a complete block twenty-one minutes after the administration of atropin. The record (Fig. 2) taken at 10.56, twenty-five minutes after the injection of atropin, shows that the block is broken and that the ventricle is responding to each auricular systole, but with a conduction period (P–R interval) of 0.4 second. Five minutes later (Fig. 3) the rate had not changed, but the conduction period was reduced to 0.22 second. The record (Fig. 4) taken thirty-six minutes after the injection of atropin shows that the rate has dropped to 80 and the conduction time to 0.2 second. In none of the subsequent records (the last was taken on December 7, just before the patient

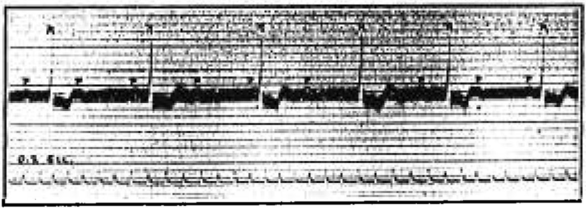

FIG. 1.—Case I. November 20, 10.51 A.M., complete block. As = 85; Vs = 46.

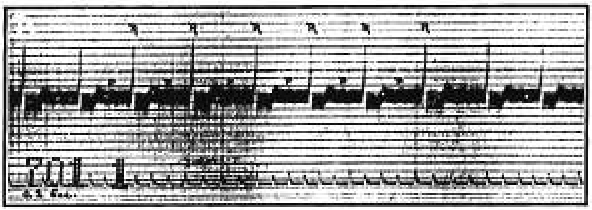

FIG. 2.—Case I. November 20, 10.56 A.M., delayed conduction; twenty-five minutes after atrophine. *P-R* interval = 0.4 second. As = 85; Vs = 85.

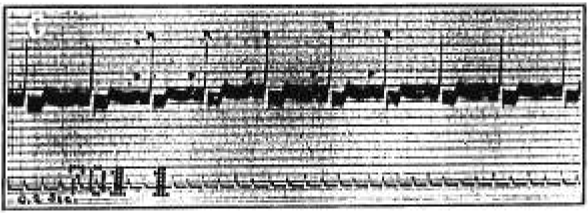

FIG. 3.—Case I. November 20, 11.01 A.M., conduction slow. *P-R* interval = 0.22 second; As = 85; Vs = 85.

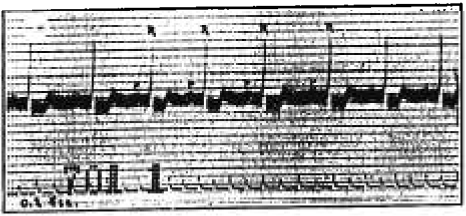

FIG. 4.—Case I. November 20, 11.06 A.M. *P-R* interval = 0.2 second; As = 80; Vs = 80.

left the hospital much improved and with his heart fairly compensated, having been without medication of any kind for two weeks) did the conduction interval measure less than 0.2 second, thus indicating a constant moderate defect in the conducting system aside from any drug influence. During the remainder of this day (November 20) the pulse was counted at half-hour intervals and was always between 85 and 95. Some time early in the morning of November 21 the pulse dropped to 44, and although no graphic records were taken, there is little reason to doubt that the block was reëstablished at this time. At 10.30 A.M. $\frac{1}{100}$ grain of atropin was given subcutaneously and the pulse gradually rose and fluctuated between 60 and 70. On November 22, at 8 A.M., the pulse was 48; atropin, $\frac{1}{150}$ grain, was given subcutaneously every three hours until November 24, when all medication was stopped. During this period and until his discharge from the hospital, on December 7, the pulse was perfectly regular and fluctuated between 75 and 85.

This case represents a type of myocardial defect which is not rare. As MacKenzie[2] pointed out some years ago, these hearts with an abnormally slow conduction are particularly susceptible to the influence of digitalis. Digitalis, according to Cushny,[3] has a two-fold effect: (1) it heightens the tone of the vagus, and (2) it modifies the functional activity of the muscle cells. Atropin apparently acts only by paralyzing the terminal fibers of the vagus.

Since in this case atropin immediately counteracted the effect of the digitalis and brought the heart back to the functional condition in which it was when uninfluenced by drugs of any kind, it is fair to conclude that here the digitalis influence was wholly a vagus effect.

Another point to be observed is that the ventricular activity is alone effected by the atropin administration; there is no evidence of change in the auricular activity. The work of Robinson and Draper[4] and Cohn and Lewis[5] indicates that nervous influences are brought to the junctional tissues more particularly by the left vagus and left sympathetic. Hence it would seem that in this case the left vagus was far more susceptible to drug influences than the right.

The rate of ventricular activity during complete block is also a subject of some interest. It is usually supposed that the ideoventricular rate in man is in the neighborhood of 30 per minute. In the case above described the ventricular rate was 46, with complete block. Among our records are eight cases of complete block in which the ventricular rate was at times over 45 per minute. May it not be that in such cases the nerve connections of the sympathetic passing to the ventricular tissues may have escaped the damage to other tissues, and so may be free to exert their influence in accelerating the liberation of stimuli produced in or below the junctional tissues.

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[2] British Med. Jour., 1905, pp. 519 and 812, also see Hewlett, Jour. Amer. Med. Assoc., 1907, xlviii, 47; Windle, Heart, 1911–12, iii, 1; Rihl, Zeitschr. f. exp. Path. u. Ther., ii, 74.

[3] Cushny, Morris, and Silberberg, Heart, 1912–13, iv, 33.

[4] Jour. Exp. Med., 1911, xiv, 227.

[5] Ibid., 1913, xviii, 739.

CASE II.—*Complete heart-block with unknown cause. No valvular defect, rate changed by atropin, but block continues.*

A. S., aged twenty-five years; trained nurse; single; American. Admitted to the Presbyterian Hospital November 10, 1913, for precordial pain. Her mother died at fifty-nine of "Bright's disease and cardiac complications." One brother and one sister died of diphtheria in childhood; otherwise the family history is good.

The patient had always led an active life, with much athletic exercise (tennis, riding, etc.). She had never been short of breath or conscious of her heart in any way. She had measles, chickenpox, whooping cough, and mumps as a child, and meningitis when five years old. When fifteen she was very sick with an attack of la grippe; at that time her tonsils were much enlarged but not inflamed. Five years ago her appendix was removed. Three years ago she had an attack of catarrhal jaundice. Formerly, but not of late, she had many headaches and occasionally an attack of "sore throat." She has never had scarlet fever, diphtheria, typhoid, malaria, rheumatism, or chorea.

Ten days before her admission she developed a severe pain in the left side of the chest in the precordial region. The pain was at first constant, somewhat relieved by pressure or by taking a deep breath, but she could not lie on the left side with comfort. The pain did not radiate. Later the pain changed to a continuous tenderness, with occasional sharp, sticking pains. For the first two days she had a severe headache. Four days before admission she vomited and had a number of loose bowel movements.

Physical Examination. A healthy, well-nourished appearing young woman with good color. The lungs are normal. There is slight tenderness in the fourth and fifth left intercostal spaces 5 cm. from the sternum. The heart impulse is not visible, but is indistinctly felt in the fifth left space 8 cm. from the midsternal line. The heart action is regular, rate 50. The sounds are of a normal quality; no interpolated sounds are heard. There are no murmurs. The pulsation of the veins of the neck is not visible. Blood-pressure: systolic, 100 mm.; diastolic, 60 mm. of mercury. Urine normal. A thorough physical examination reveals no other abnormality. The Wassermann test is negative. When examined with the fluoroscope an independent rhythm of the auricles and ventricles is distinctly made out.

Numerous electrocardiographic records confirmed the diagnosis of complete heart-block. Neither the auricles nor ventricles contracted in a perfect rhythm, and their activities were entirely independent of one another. On various examinations the rates varied, As = 65 to 75; Vs = 34 to 48.

On November 18 observations were made following the subcutaneous administration of $\frac{1}{50}$ grain of atropin, when the following data were obtained:

3.40 P.M.	As = 75.	Vs = 48, complete block (see Fig. 5).
3.42 P.M.	Atropin $\frac{1}{50}$ grain hypodermically.	
3.47 P.M.		Vs = 48
3.52 P.M.		Vs = 48
3.57 P.M.		Vs = 50
4.02 P.M.		Vs = 54
4.07 P.M.	As = 90.	Vs = 52, complete block (Fig. 6).
4.10 P.M.	As = 100.	Vs = 60, complete block (Fig. 7).
4.12 P.M.	As = 100.	Vs = 60, complete block (Fig. 8).

All the records of this case were taken by Lead II (right arm and left leg). It is evident that the auricles and ventricles are dissociated in all of the records; it is also clear that in any given record neither auricles nor ventricles have a perfectly rhythmic activity of their own, both the auricular cycles and the ventricular cycles varying in length to a considerable degree.

When we come to examine the results of the administration of atropin we find (1) the auricular rate is increased from 75 to 100; (2) the ventricular rate is increased from 48 to 60; (3) the block between the auricle and ventricle is complete and persists in spite of the atropin; (4) the independent arrhythmias of the auricles and of the ventricles disappear after the full effect of the atropin.

The patient's pain gradually wore away after a few days' rest in bed; she left the hospital for a few weeks of vacation and then returned to the strenuous duties of an active hospital nurse, at which she is at present occupied, with no inconvenience or discomfort. Her present pulse is between 40 and 50 and shows the same characters as described before the administration of atropin.

The persistence of the block in this case leads us to conclude that the conduction system is functionally severed. Even on the removal of all nervous influences with atropin the bundle of His is unable to convey impulses from the auricle to the ventricle. Notwithstanding this fact it is quite evident that the activities of the ventricle are modified by vagus influences, for when these were removed the ventricular rate increased from 48 to 60. Furthermore, when the vagus influence is taken away the ventricle becomes rhythmic, it is generally accepted that the so-called *sinus arrhythmias* are due to extrinsic nerve influences; by analogy it seems not improbable that in this case the arrhythmia of the independently acting ventricle is due to a similar cause. On these grounds it would appear that whatever its nature the lesion causing the block has left uninjured structures which still allow the vagus to modify

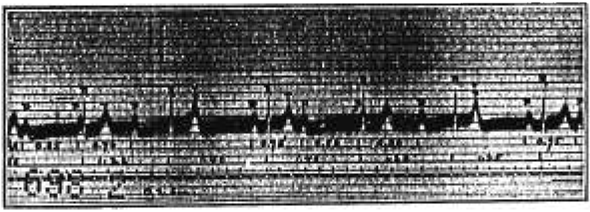

Fig. 5.—Case II. November 18, 3.40 P.M., before atropine; complete block. As = 75 arrhythmic; Vs = 48 arrhythmic.

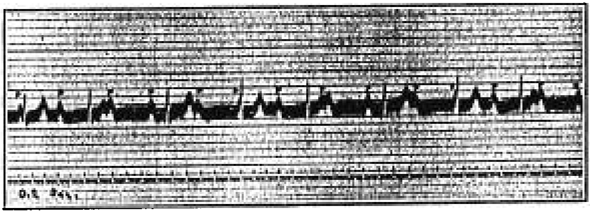

Fig. 6.—Case II. November 18, 4.07 P.M., twenty-five minutes after atropine; complete block. As = 90; Vs = 52.

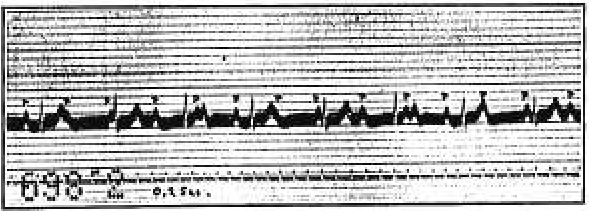

Fig. 7.—Case II. November 18, 4.10 P.M.; complete block. As = 100; Vs = 60.

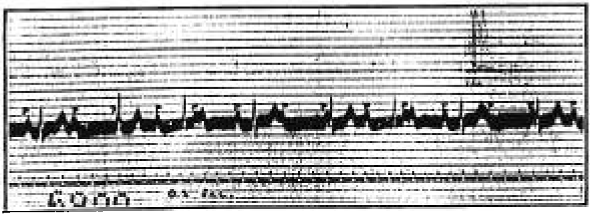

Fig. 8.—Case II. November 18, 4.12 P.M., complete block. As = 100 rhythmic; Vs = 60 rhythmic.

the activities of the ventricle. I have observed two other cases of complete block which responded to atropin in a manner similar to the above; although the block was not broken the ventricular rate was measurably increased.

CASE III.—*Decompensated heart; auricular fibrillation; atropin increases rate without removing block.*

W. H. R., aged forty-eight years, letter-carrier; American. Admitted to the Presbyterian Hospital, September 27, 1913, complaining of dyspnea. His mother died at thirty-nine of "heart disease." He has never had scarlet fever, diphtheria, typhoid, malaria, rheumatism, chorea, or syphilis. He had three attacks of gonorrhea. He had an attack of pneumonia ten years ago. He does not use alcohol or tobacco. Twelve years ago he was told that he had a large heart. Four years ago he "collapsed" while at work; he did not lose consciousness, but following this was confined to the house for two weeks suffering from weakness. For two and a half years he had had gradually increasing dyspnea on exertion. Twenty months before admission he fainted at the end of a day's work and was in the house five weeks from weakness. Nine months ago he stopped work on account of weakness and shortness of breath. He first noticed swelling of the legs four months before admission. For two months he has been obliged to sit up in bed to sleep. The abdomen had become much enlarged and the fluid was drawn off four weeks before admission. Examination on September 27: Cyanotic and dyspneic. There was a right hydrothorax, extensive abdominal ascites, and edema of the legs. The liver extended from the fourth space to 6 cm. below the costal margin in the right midclavicular line; there was no pulsation. The urine averaged a daily output of 600 c.c., with a trace of albumin and a few casts. The sulphophenolphthalein output for two hours was 58 per cent. The blood showed a mild grade of secondary anemia; the Wassermann reaction was negative at this time and on several subsequent examinations.

The apex of the heart was in the seventh space 18 cm. to the left of the midsternal line; no dulness to the right of the sternum. The action was slow and irregular; the rate about 40 per minute. The sounds were somewhat impure and muffled, the aortic second louder than the pulmonic second. No murmurs could be heard. The blood-pressure indicated that the majority of the cardiac contractions were audible below the brachial cuff at 154 mm. of mercury; the diastolic pressure was estimated at 96 mm. All of the cardiac contractions heard at the apex could not be felt in the radial artery. The character of the irregularity as determined by auscultation at the apex was as follows: for short periods the heart beat was slow and perfectly rhythmic, with a pause between the systoles of about one and a half seconds—this rhythm was interrupted at irregular intervals by an extra beat which quickly

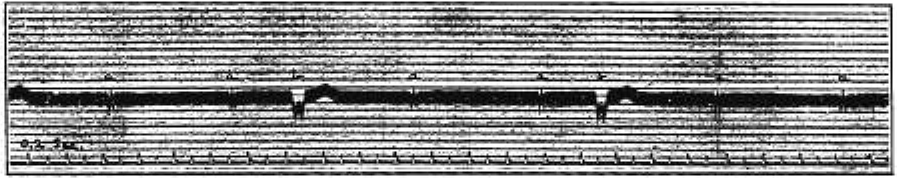

FIG. 9.—Case III. October 11, 1913. Lead I. Ventricular complexes, types *a* and *b*. Vs = 60.

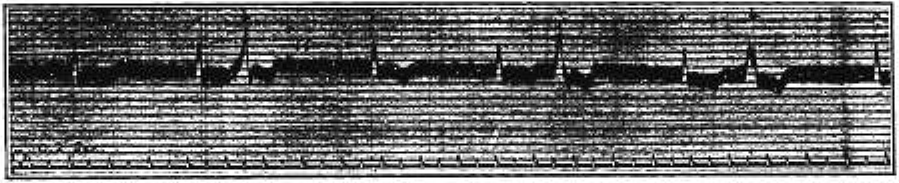

FIG. 10.—Case III. October 11, 1913. Lead II. Ventricular complexes, types *a* and *b*.

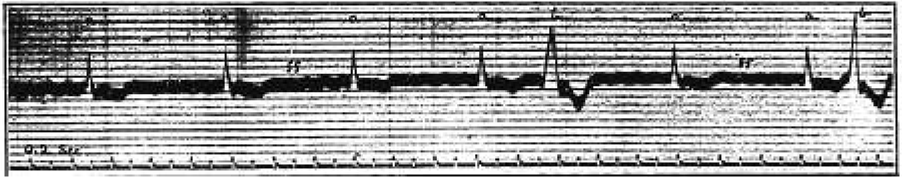

FIG. 11.—Case III. October 11, 1913. Lead III. *ff* = auricular fibrillary waves; ventricular complexes, types *a* and *b*.

succeeded one of the systoles of the slow rhythmic beats and was followed by a pause equal to the intersystolic pauses of the slow rhythm; the sound of the extra beats was less distinct than the others, but both first and second sounds could be heard; the impression was one of a slow rhythmic heart occasionally interrupted by extrasystoles. An electrocardiogram (Figs. 9, 10, 11) taken at this time showed an auricular fibrillation and slow ventricular activity perfectly rhythmic except when the rhythm was interrupted by an extrasystole arising from the left ventricle.[6] The rhythmic beats were separated by an interval of 1.4 seconds and the heart rate, if the rhythm had been undisturbed by the extrasystolic contractions for a period of a minute would have been 36 per minute. The extrasystolic contractions occurred always 0.5 seconds after a beat of the rhythmic series, and each was followed by a pause of 1.4 seconds before the beats of the regular rhythm were resumed.

Briefly the subsequent course was as follows: Removal of the fluid from the chest and abdomen was followed by a period of gradual improvement until October 27, when he suddenly developed a paralysis of the muscles of the right side of the face and a complete motor aphasia, after this he gradually improved and left the hospital the middle of November. He was readmitted December 26 with compensation again broken, ascites, and hydrothorax.

At this time the heart sounds were of poor quality: The pulmonic was louder than the aortic second sound, and there was occasionally heard a short diastolic murmur from the second left space to the apex where it had a musical quality. The pulse rate was between 28 and 32. On ausculation it was evident that the number of extrasystoles had considerably increased over the condition as observed during his former stay in the hospital. In addition to the single extrasystoles following the rhythmic beats as above described a second extrasystole could frequently be detected, of which the second sound was sometimes heard and was at times inaudible. Practically none of the extrasystoles was forcible enough to reach the wrist.

The electrocardiograms on December 29 are shown in Figs. 12, 13, 14; the ventricular rate is 60, the auricle is fibrillating. This shows complexes of the normal ventricular form (type *a*), a second type (*b*) whose form indicates the starting-point to be a portion of the left ventricle near the base, and more rarely a third type (*c*), having its origin in the apical portion of the left ventricle.[7] The time relations of these waves of ventricular activity are quite constant, the ectopic complexes following the preceding complex at an interval of 0.6 second; the normal complex (type *a*) is always preceded by a pause of 1.4 seconds.

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[6] Rothberger and Winterberg, Arch. f. des ges. Physiol., 1913, cliv, 571.

[7] Ibid.

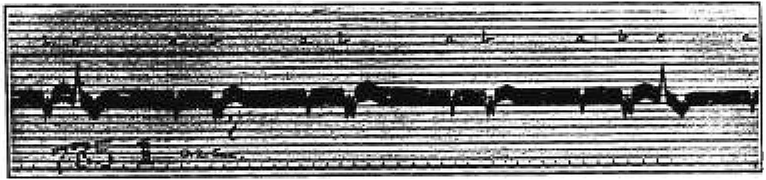

FIG. 12.—Case III. December 29, 1913. Lead I. Ventricular complexes, types *a*, *b* and *c*. Vs = 60.

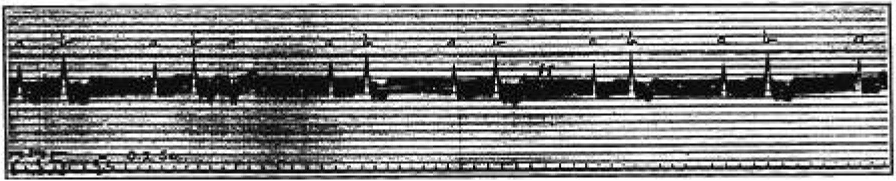

FIG. 13—Case III. December 29, 1913. Lead II. Ventricular complexes, types *a*, *b* and *c*. *ff* = auricular fibrillary waves.

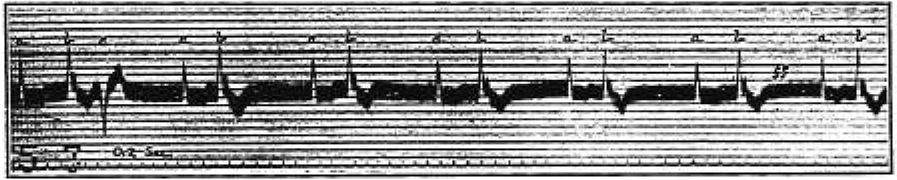

FIG. 14.—Case III. December 29, 1913. 5.35 P.M. Lead III. Ventricular complexes, types *a*, *b* and *c*. *ff* = auricular fibrillary waves.

A few minutes after this record (Figs. 12, 13, 14) was taken $\frac{1}{30}$ grain of atropin was given hypodermically; the effect may be seen in Figs. 15, 16, 17, 18, all taken by Lead III. The changes which occurred may be tabulated as follows:

	Rate.			Ventricular complexes.					
Time.	Radial.	Ventricle.		Type (*a*)		Type (*b*)		Type (*c*)	Figure.
5.35 P.M.	28	60	=	28	+	28	+	4	14
5.41 P.M.	..	(Atropin $\frac{1}{30}$ grain hypodermically)							
5.48 P.M.	32	68	=	32	+	32	+	4	
5.51 P.M.	40	68	=	40	+	27	+	1	
5.54 P.M.	..	60		..		..		..	15
6.00 P.M.	..	75		..		..		..	16
6.01 P.M.	68	76	=	68	+	8			
6.03 P.M.	..	77							
6.05 P.M.	72	76	=	72	+	4			
6.07 P.M.	..	75		..		..		..	17
6.11 P.M.	..	76							
6.15 P.M.	..	76		..		..		..	18

It is to be noted that the auricular fibrillation persists throughout the whole period, the ventricular contractions are increased from 60 to 77 in twenty-two minutes, at the rapid rates nearly all of the ventricular complexes are of the normal form (type *a*), none of type *c* persist, and when type *b* appears it occurs a little earlier than is usual for the normal complexes (type *a*), and is followed by a pause which is completely compensatory (measured by the rhythm of the complexes of type *a*). With the exception of the occasional ectopic beats the ventricle contracts in a perfectly rhythmic manner. The contractions corresponding to the normal complexes (type *a*) all, and these alone, are forcible enough to furnish arterial waves which are palpable in the radial artery.

After the administration of atropin on December 29 the radial pulse remained above 60 for about eight hours and then fell to 35. Subsequently atropin was given regularly ($\frac{1}{100}$ grain t. i. d. hypodermically) for periods of several days; by this means the pulse could be kept between 50 and 60. At these times the patient felt better until dryness of the mouth and paralysis of accommodation caused its discontinuance. At the present time (May, 1914) his general condition has considerably improved, although the rate and rhythm of the heart are much the same as on his admission on December 26.

This case represents a type which was first described by MacKenzie[8] under the term "nodal bradycardia;" at the time of this early description he did not recognize that the auricle was fibrillating, but this was pointed out by Lewis[9] at a later time based on electrocardiographic records of one of the same series of cases which MacKenzie had studied. Similar cases have since been reported by Lewis,[10] Cohn,[11] Draper,[12] and others. The presence

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[8] Heart, 1909-10, i, 33.

[9] Ibid., 1909-10, i, 351.

[10] Heart, 1911-12, iv, 15.

[11] Ibid., 1911-12, iii, 23.

[12] Ibid., 1911-12, iii, 13.

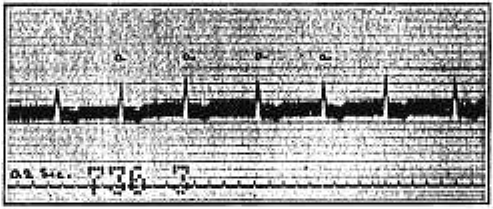

Fig. 15.—Case III. December 29, 1913, 5.54 p.m., thirteen minutes after atropine. Lead III. Vs. = 60.

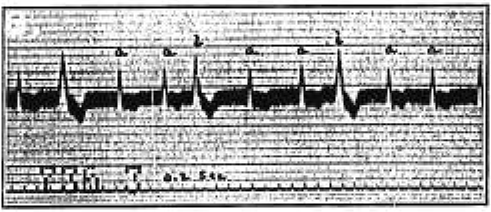

Fig. 16.—Case III. December 29, 6 p.m. Lead III. Vs = 75.

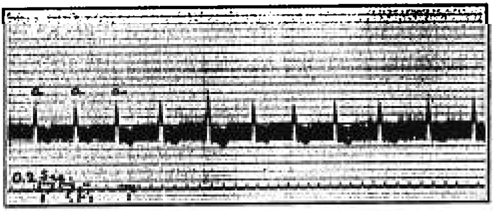

Fig. 17.—Case III. December 29, 6.07 p.m. Lead III. Vs = 75.

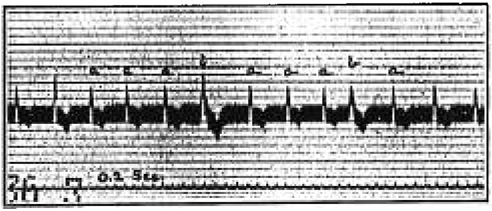

Fig. 18.—Case III. December 29, 6.15 p.m. Lead III. Vs = 76.

of extrasystoles and their increasing frequency indicates the myocardial damage which is progressive; the evidence goes to show that at first one point and later two points in the left ventricle have become excessively irritable. The inefficiency of these beats having an ectopic origin is evidenced by the fact that only a few of these are capable of causing a wave which can be detected in the radial artery, yet it is clear that these same contractions are exhausting to the cardiac tissues, since the pause after the ectopic beat is of the same length as the interval between two beats of the undisturbed ideoventricular rhythm.

We have no reason to suppose in this case that we have removed the block by means of atropin. Except for ventricular extrasystoles the heart is rhythmic. We have simply removed the vagus control of the ventricle. The ventricle is working fairly rapidly (76), but in perfect independence of the irregular fibrillary impulses sent down from the auricles. (This activity reminds one very much of cases of auricular fibrillation which are frequently seen in which after a course of digitalis the pulse becomes comparatively slow—presumably a partial block has been established—and regular at a rate between 70 and 80, although the auricles continue their fibrillary activity. In these cases, however, a dose of atropin not only causes the ventricle to beat much faster, but the artificial digitalis-block being removed the ventricle again beats with complete irregularity.)

In the case here reported the administration of atropin caused an increase in the ventricular rate from 60 to 77 in twenty-two minutes. More impressive than this, however, is the fact that under this influence the number of contractions of type *a* (which are the normal ventricular contractions for this heart) increase from 28 to 72 and the beats of an ectopic origin almost disappear (falling from 32 to 4), this seems to indicate that the removal of vagus influence has permitted the normal pacemaker of this ventricle to increase its rate of production of stimuli over 150 per cent. and thus to supplant almost completely the impulses set free from the ectopic foci.

Here again if we are correct in our interpretation we are forced to the conclusion that in spite of the auriculoventricular block the vagus has no inconsiderable control over independent ventricular activity. We have seen one other case of auricular fibrillation and block with occasional ectopic ventricular contractions which we have studied electrocardiographically with and without atropin; the atropin effect was quite similar to the case here described.

SUMMARY. There are reported in detail three cases of heart-block representing three distinct types:

1. Complete block following digitalis.
2. Complete block with normal auricular activity.
3. Complete block with fibrillating auricles.

Other cases are referred to in support of the evidence presented in detail from these cases.

The effect of atropin administration was studied in each of these cases. In the first case (digitalis-block) the dissociation was removed; the evidence of the modification of ventricular activity was considerable. Evidence of the modification of auricular activity was negligible

In the second case (block with normal auricular activity) the block was not removed but the ventricles were much more affected than the auricles. In the third case (block with fibrillating auricles) atropin did not destroy the block but allowed the ideoventricular rate to be greatly increased.

If we are right in assuming that atropin acts only by paralyzing the terminal filaments of the vagus it is evident that in each of the cases here described the vagus had a very important influence in controlling the ventricle, either through the junctional tissues or directly on the ventricular tissue.[13]

Case I emphasizes the fact that the vagus influence is sufficient to establish a block between the auricle and the ventricle.

Cases II and III indicate that even in the presence of a lesion sufficient to cause complete dissociation between the auricles and ventricles the nervous connections may remain in a condition of functional activity and thus continue to modify the ventricular rate and rhythm.

It is suggested that in certain cases of block the sympathetic nerve connections may escape damage and may thus be capable of exerting a direct accelerator influence on the ventricle.

The disappearance of the extrasystoles associated with an increase in the activity of the predominant pacemaker is illustrated.

For the privelege of studying a number of these cases I am indebted to the courtesy of my colleague, Professor Longcope, whose hearty coöperation while the patients were on his service in the Presbyterian Hospital has made these observations possible.

ACONITE AS A VASODILATOR.

By William Hanna Thomson, M.D.,
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One of the important questions in modern therapeutics is how to regulate arterial blood-pressure, particularly when it rises above normal. Morbid conditions due to high pressure are very numer-

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[13] For further views of the control of the ventricle by the vagus, see Ritchie, Quart. Jour. Med., 1912-13, vi, 62.